THE VETERINARY JOURNAL

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EDITORIAL

THE PROFESSION IN AUSTRALIA

It is probably unique for the editorial column of a great newspaper to be devoted to an appreciation of the veterinary profession. Such has been the case recently in a great Australian paper. We are certain that the members of the profession in all other parts of the Empire will be delighted to know that the work of their Australian colleagues is so greatly appreciated. The occasion which led to the editorial was the annual conference of the Australian Veterinary Association in Sydney. The conference was also addressed by the Minister of Agriculture, Mr. W. F. Dunn, who paid eloquent tribute to veterinarians generally in their war and pre-war work for the livestock industry of Australia.

We feel that these appreciations of the value of the veterinary surgeon to the community should be brought to the notice of all those who work in the fields of veterinary science and animal husbandry throughout the Empire, and it is with pride that we reprint the following extracts from *Country Life*, the Australian stock and station journal (29/10/1943) :—

GUARDIANS OF ANIMAL HEALTH

[Editorial from *Country Life*]

" Bodyguard of Australia's livestock industry embracing the first (wool) and fourth (meat) of our normal exports, a hundred veterinary scientists met in Sydney this week under the ægis and in membership of the Australian Veterinary Association.

" The five score men constituted a body with various lengths of service to the pastoral, farming, pig-raising, poultry-breeding and kindred animal industries ranging from two score years to the fledgling; from the Director of the Animal Health Division of the Council of Scientific and Industrial Research, to the Dean of the Faculty of Veterinary Science, to the private practitioner, Commonwealth and State veterinary leaders and officers, and including research work of the widest possible variety. They represented, too, every State in the Commonwealth.

" If Sydney knew what it owed to those men it would have extended them

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the freedom of the city. But Sydney knew them not—the country knows them better, but the whole Commonwealth should join in praise and approval of their never-tiring, behind scenes, expert and selfless tasks, on behalf of food production.

"These men deserve the best pastoral and allied industries can do to co-operate with them. Some of them are getting that—others are developing every possible avenue of approach to secure such confidence and trust, more still are urgently needed by both veterinary and pastoral circles to join in the work of maintaining animal health and animal food supplies for the nation now in war, and in future, in peace.

"Veterinarians, we are proud to state, comprise in Australia a body militant. Their Commonwealth-wide organisation covering and linking together, through branches, all States of Australia, reflects the breadth of vision and extensiveness of operations they represent.

"Few bodies or individuals are less publicised yet by the graziers of this State, especially there is a lively appreciation of what the veterinarians have done to not only maintain livestock health, but to keep disease out of this large but fortunate country.

"We know that the way of life of veterinarians becomes that of the researcher, spending nights in 'nightly days' painstakingly and persistently seeking approaches to specific conditions; this they have in common, the private practitioner and the State official. Instance of their co-operation and mutual contact is the fixation test by which pleuro-pneumonia can be diagnosed by blood test of the apparently healthy beast—a tribute to the work of the C.S.I.R.

"And, again, we have the instance of black disease in swampy country being now controlled—in this case by a vaccine evolved and, if necessary, demonstrated by the same set of officers.

"Most gratifying indeed was it to find at the Conference that the problems of the livestock industry were viewed with the interest and buoyancy of youth, even by men who have grown old in the service of the community and who would be excusable if their outlook had consequently become a little tinctured by cynicism.

"Most of these men were graduates of the School of Veterinary Science, within the Sydney University, where the conference was held—and their discussions, specific and broadly national, were not only a credit to all associated with them, but to P.P. Boards, who have championed the Veterinary School."

FINE TRIBUTE TO VETERINARIANS

[At opening of Annual Conference of the Australian Veterinary Association]

In the course of his address the Minister said:

"Prior to the commencement of this war, there was a steadily growing appreciation of the important place which the veterinary profession held in the economic well-being of our community. The events of the early stages of the war did not materially alter the position, as it was not then apparent that the problem of food would become one of outstanding importance. The changing situation in the last year or so has, however, placed food production, especially food of animal origin, in the forefront of our problems, and as a result the

value of the services which your profession may render to the country has increased beyond anything previously understood.

"The old idea of the veterinarian as one who merely treated sick animals has died hard, but it is now thoroughly and permanently dead. Your services in the production of food of animal origin covers a far wider field. It lies firstly in the prevention of disease, whether disease is due to parasites or bacteria, or to nutritional and breeding problems, and secondly, in the stepping up of production through improved methods of animal husbandry.

"It is fully realised nowadays that health and production are indivisible, and that without good health high production cannot be secured. Therefore, your work lies at the basis of the drive now being undertaken to secure ever increasing quantities of meat, dairy produce and eggs.

"Although I stress this aspect of your work as being the one which is of first national importance at the present time, I do not forget that some of you are busily occupied in treating those animals to which we ascribe so much sentimental value as our domestic pets. Even in war time one cannot deny the value of sentimental feeling as tending to uphold morale.

"I know that many of you have been disappointed that in the present war the changed conditions of armament have led to a great decrease in the demand for veterinarians and you are no longer able to repeat the fine work done by the Army Veterinary Corps in the last war. Still, I understand that some of your colleagues are serving in the Armed Forces, not only in the professional aspect, but in combatant units, and that some of you in your civil capacity are engaged in important work in connection with the meat and milk supplies for our own and allied troops. In that respect they must be considered as performing work of national value, since the healthfulness of the food of the troops is a matter of outstanding importance.

University Farm

"As we are meeting in the University, it is right that I should make some reference to the education of the veterinary surgeon. It is in keeping with the moving spirit of the times that the training given here has an increasing bias towards animal husbandry, and I feel we owe much to the enlightened outlook of the McGarvie Smith Institute in providing funds which have enabled the University to secure the farm at Badgery's Creek, at which the students spend an appreciable portion of their time in carrying out actual practical work with livestock. I realise how the difficulties of matters associated with the war have increased the burden on those remaining. It is a pity that this, the only school now functioning in Australia, should not be fully staffed.

"It appears likely that the number of students entering the school will increase, and this makes it more than ever necessary that staff and facilities should be adequate.

"I am advised that much is required in the way of both staff and equipment if the standard of this school is to be maintained and improved. No matter what standard is attained, there is always room for improvement, and I would always be prepared to consider carefully any proposals which might be put forward to me towards that aim. It might not, of course, be possible for me

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to do much myself, but it might be that I could advance your case with those who could materially assist.

"The veterinary profession in Australia is not numerically very strong, and in that respect the position compares unfavourably with most stock-raising countries. The position must be changed if this country is to develop its livestock activities on sound lines; if our export trade in animal products is to be maintained or increased and if the health of our population is to be secured.

"It may be a new idea to some that the veterinarian has a part to play in maintaining human health, but there are many ways in which he does so. He is responsible for taking such steps as will keep rabies and glanders and other serious diseases of man out of the country; he is responsible and should be increasingly responsible for dealing with those milk-borne diseases such as tuberculosis which attack man; and through safeguarding the production of milk and other protective foods he may have a profound influence on the health of the community.

"I notice that the veterinary surgeons can now, through the Australian Veterinary Association, speak with a united voice, and that you have wisely incorporated your State associations as divisions of the Australian Veterinary Association. In so doing I think you have acted wisely. I would commend to every veterinarian in Australia the idea that he should actively support his association."

General Articles

CYANIDE POISONING AND ITS TREATMENT

By G. D. SHEARER and K. C. SELLERS,
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THE most frequent cause of poisoning of farm animals by HCN in this country is by the consumption of foodstuff containing much cyanogenetic glucoside. Many of the leguminoseæ, in particular Java or Rangoon beans (*Phæseolus lunatus*), certain of the gramineæ, sorghum and maize and among the linaceæ, common flax (*Linum usitatissimum*), from which linseed cake is prepared, and purging flax (*Linum catharticum*), contain these glucosides which are decomposed with liberation of HCN by the enzyme which almost invariably accompanies them in the plant. The glucoside and enzyme are in themselves harmless to the animal, and only when the glucoside is decomposed with liberation of HCN can its poisonous properties be manifested. Although some plants containing cyanogenetic glucosides may not contain enzyme for their decomposition, Seddon and King (1930) have found that sufficient of the enzyme to decompose the majority of glucosides is contained in the ingesta of sheep after it has eaten green forage. After mastication of the cyanogenetic food by the animal the pulped mass at body temperature is in the most favourable condition for fermentation to take place and the liberation of HCN is therefore rapid in the rumen. As pointed out by Rimington (1932), when a fresh plant or plant powder is consumed the conditions are peculiar in that the cyanogenetic glucoside and the appropriate enzyme are enclosed within the plant cell, the

proteins of which constitute a very efficient buffer system and interaction between the enzyme and glucoside takes place at a pH approaching the optimum before the mass becomes permeated with the acid gastric juice.

Care should be exercised before it is concluded that the death of an animal is due to the consumption of foodstuff containing dangerous amounts of cyanide unless chemical evidence supports this view or the classical symptoms of cyanide poisoning are evident. Bunyea (1934) considers that if a foodstuff contains as little as 0.02 per cent. potential HCN and if the animal consumes it all rapidly, 5 lb. would prove fatal to a horse or cow and 1.25 lb. would kill a sheep. But he also points out that cattle fed on a high starch diet develop a resistance to cyanide poisoning. Worden (1940) gives an account of work done by Lander (1910) and of Tocher (1935) on the feeding of linseed cake containing significant quantities of cyanogenetic glucoside. In one case Lander fed 5 lb. per day of linseed cake containing 0.025 per cent. of HCN to a six-month-old heifer for twenty days without harm. Rogers and Boyd (1936) fed cattle on Sudan grass at various stages of growth without producing any effect, and conclude that death following the eating of cyanophoric plants may be due to other factors than cyanide alone. However, we find it difficult to believe that the repeated ingestion by the animal organism of small amounts of cyanide is not without harm to the animal, especially when we take into account the work of Weston Hurst (1942), using sheep and monkeys, and of Ferraro (1933), using cats and monkeys, who both observed demyelination following the repeated injection of toxic but sub-lethal amounts of KCN.

Of recent years much attention has been paid in America and in the Antipodes to the question of poisoning of farm stock by the consumption of cyanogenetic foodstuffs, and as a result of work done at the Bureau of Animal Industry of the American Department of Agriculture a successful remedy has been evolved. Bunyea (1935) has given a résumé of the work done by himself and his colleagues.

The symptoms observed in fatal cases of poisoning of cattle and sheep were increase in depth and rapidity of respiration, convulsions of varying degree and duration, opisthotonus and jerky movements of the eyeball. In cattle, the jugular vein was prominent and engorged and the blood of a bright red colour. The most characteristic lesions observed on post-mortem examination were darkened musculature, congestion and hæmorrhage of the lungs, petechiæ on the tracheal and bronchial mucosa and a frothy bloody discharge from the mouth. He also gives an account of the work done on the use of various remedies in which they used methylene blue, sodium tetrathionate, sodium thiosulphate and sodium nitrite, none of which, by itself, was found to be very effective, but a combination of sodium thiosulphate and sodium nitrite proved to be very successful. The remedies were given intravenously or intraperitoneally. The M.L.D. of KCN, calculated as HCN, was found to be 2.042 mgs. per kilo body weight for cattle and 2.315 mgs. per kilo body weight for sheep. Cattle which had received 2 M.L.D.s *per os.* of KCN recovered when given 10 ml. 10 per cent. $NaNO_2$ and 20 ml. 10 per cent. $Na_2S_2O_3$ intraperitoneally. The most effective dose of the remedy used by Bunyea, Couch and Clawson (1934) was about 1 g. $NaNO_2$ and 2 g. $Na_2S_2O_3$ i.p. in 15 ml. water for a 30-40 kg.

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sheep; 2 g. $NaNO_2$ is the largest dose which should be given to such a sheep. In Australia, a solution of 3 g. $NaNO_2$ and 15 g. $Na_2S_2O_3$ in 20 ml. water administered subcutaneously has been used with success in the treatment of cattle poisoned as a result of grazing on Sudan grass [Rose (1941)], and when any animals show symptoms of poisoning all the members of the herd are inoculated as a further preventative. In animals showing tympany the use of the trocar and canula to relieve intra-abdominal pressure is an essential precaution. The remedy has also been used with success in the treatment of human cases of cyanide poisoning. A young girl who had taken 5 g. KCN by mouth was given one ampoule of amyl nitrite and 20 ml. of 30 per cent. $Na_2S_2O_3$ intravenously; she was given a further 20 ml. of 20 per cent. $NaNO_2$ but, as a result, suffered from severe cyanosis due to the too liberal use of nitrite. The following day, however, she had recovered. Another woman had taken about 2 g. KCN and was found comatose. She was given an aqueous solution containing 0.75 g. $NaNO_2$ and 12 g. $Na_2S_2O_3$ intravenously and made an uneventful recovery [Viana, Cagnoli and Cendan (1934)].

TABLE I—RABBITS

No.	*Wt. Kg.*	*Dose*	*Symptoms*	*Result*
A.58	1.3	0.9 ml. 1% KCN = 2.9 mg. HCN/kg.	Increased respiration	Lived
A.63	1.2	0.85 ml. 1% KCN = 2.9 mg. HCN/kg.	Increased respiration	Lived
A.60	2.22	1.3 ml. 1% KCN = 2.45 mg. HCN/kg.	3.5 p.m. dosed 3.10 stretched out, convulsions 3.15 moribund, resps. 5/15 secs. 3.25 dead. Pupils after death contracted	Died
A.67	1.87	1.3 ml. 1% KCN = 2.9 mg. HCN/kg.	3.10 p.m. dosed 3.15 stretched out, convulsions, scouring 3.20 moribund, resps. 5/20 secs. 3.25 dead. Pupils after death contracted	Died
A.2	2.2	1.9 ml. 1% KCN = 2.9 mg. HCN/kg.	12.20 p.m. dosed 12.25 stretched out. 1 c.c. nitrite and thiosulphate i.p. 12.30 severe convulsions 12.35 severe convulsions, resps. 10/10 secs. 12.40 resps. 10/7 secs. 12.45 sat up 2.0 seemingly recovered Next day recovered	Lived
392	2.9	1.7 ml. 1% KCN = 2.44 mg. HCN/kg.	12.5 p.m. dosed 12.10 stretched out. 1.5 c.c. nitrite and thiosulphate i.p. 12.20 sitting up 12.50 almost normal 5.0 apparently recovered	Lived
82	2.2	1.5 ml. 1% KCN = 2.9 mg. HCN/kg.	11.57 a.m. dosed 12.10 p.m. stretched out 12.20 dead	Died
C.84	2.3	1.4 ml. 1% KCN = 2.5 mg. HCN/kg.	12 noon dosed 12.27 p.m. eye reflex gone, lying down, whisker reflex reduced 12.30 air hunger, resps. 10/10 secs. 12.35 convulsions 12.45 moribund, resps. 10/12 secs. 12.50 1.5 ml. nitrite and thiosulphate, eye reflex better, much improved generally 2.25 apparently recovered	Lived

TABLE II—SHEEP

No.	*Wt.*	*Dose*	*Symptoms*	*Result*
1 Ram about 5 months	6 st.	By mouth 5.3 mg. KCN/kg. 2.2 mg. HCN/kg.	3.8 p.m. dosed *per os.* 3.12 deepened and increased resps., profuse salivation 3.15 salivation reduced 3.17 trembling 3.20 much deepened respirations 3.30 slightly improved 3.35 still trembling somewhat more violently 3.42 deep breathing 3.45 observations stopped 4.45 apparently recovered and eating; resps. normal	Lived
Sheep 2	57 lb. 5 mths. ewe lamb	5.3 mg. KCN/kg. 2.2 mg. HCN/kg.	9.10 a.m. dosed by mouth 9.12 breathing deeply 9.15 down 9.20 up, trembling 9.40 trembling reduced	Lived
Ram used before	6 st.	8.43 mg. KCN/kg. 3.49 mg. HCN/kg.	11.25 a.m. dosed 11.28 down, breathing deeply 11.55 occasional tetanic spasms 12.35 p.m. found dead	Died
Ram 2	4 st. 3 lb.	8.43 mg. KCN/kg. 3.5 mg. HCN/kg.	11.35 a.m. dosed 11.37 down, breathing almost stopped 11.40 given 12 c.c. thiosulphate and nitrite i.p. Resps. stim. artificially. Remained down 11.55 not much improved 12.35 p.m. up and much improved. Recovered	Lived
Ewe A	13 st. 5 lb.	5.56 mg. KCN/kg. 2.3 mg. HCN/kg.	2.45 p.m. dosed 2.46 breathing deeply and increased 2.50 15 c.c. thio. and nitrite i.p. 2.53 much improved, champing 3.10 down, quiet 3.34 still quiet 4.45 up and eating Next day turned out to grass	Lived
Ewe B	12 st. 5 lb.	5.56 mg. KCN/kg. 2.3 mg. HCN/kg.	2.31 p.m. dosed 2.32 dyspnœa 2.35 down 2.37 deepened breathing, cyanosis 2.47 15 c.c. thio. and nitrite i.p. 2.55 quivering and champing 3.0 increased trembling, down 3.7 2 c.c. 0.6 per cent. meth. blue, i.p.*, marked cyanosis 3.35 somewhat improved 4.45 still down but improvement maintained. Next day recovered and turned out to grass	Lived

* As this animal seemed to have developed cyanosis to a dangerous degree, methylene blue was injected i.p. as Scott (1941) found methylene blue to be valuable in the treatment of nitrite poisoning.

Morbid anatomy of dead sheep. Blood brownish in colour, reddening on exposure to air. Rumen much distended. Intestines apparently normal. The liver was somewhat congested and friable. The kidneys were congested and the bladder empty. The lungs were acutely congested with extravasation of blood into the alveoli and bronchioles, more especially in the lying side. Blood-stained froth in the trachea. Hæmorrhages in the myocardium, clot in the left ventricle. Mouth and nose contained blood-stained froth.

The foregoing tables give an account of the experiments conducted on rabbits and sheep and the composition of the remedy when administered was 1 g. $NaNO_2$ and 2 g. $Na_2S_2O_3$ in 15 ml. water.

Discussion

Most forms of animal and plant life which use oxygen are rapidly destroyed

by cyanide, whose poisonous property is due predominantly to its interference with the oxidative enzymes of tissues. Death in animals is due to a form of internal asphyxia or histotoxic anoxia. Cyanide is not a blood poison but a cell poison, so that it seems unlikely that cyanide reacts directly with hæmoglobin in the circulating blood. It is known, however, that cyanide can easily combine with oxidised hæmoglobin (methæmoglobin).

In this method of treatment of cyanide poisoning by injection of nitrite and thiosulphate the nitrite oxidises the hæmoglobin within the corpuscles to methæmoglobin (Keilin and Mann, 1941), and so facilitates the conversion of free cyanides circulating in the blood-stream to cyanomethæmoglobin. This complex is slowly decomposed with liberation of HCN, which combines with the thiosulphate, which has also been injected, and forms the harmless thiocyanate. But it is not certain at present to what extent the bright red colour of the blood, which is a feature in fatal cases of cyanide poisoning, is due to the formation of cyanmethæmoglobin (Vlès, 1920). It has been shown by Evans (1919) that the oxygen of the blood is more readily dissociable in cyanide poisoning and the alkali reserve reduced, which would account for the greater stability of oxyhæmoglobin and the red colour of the blood. On Sir Joseph Barcroft's suggestion and with his collaboration, a rabbit was poisoned by injection into the jugular vein of a solution of KCN at the rate of 5.1 mgs. per kilo body weight. A sample of blood was withdrawn before injection and another when the animal was moribund. These samples of blood, of the same visual colour, were examined spectroscopically by Professor Keilin and Dr. Mann, of the Molteno Institute for Parasitology, but cyanmethæmoglobin was found to be absent.

This supports the fact that the cause of death from cyanide poisoning is due to interference with the oxidative enzymes of the tissues, in particular cytochrome and cytochrome oxidase which, owing to the reversible inactivation of cytochrome oxidase, would account for the surprising recovery of animals poisoned by cyanide, as reported by Barcroft (1931) and by Worden (1940).

Summary

A short account is given of the occurrence of cyanide poisoning of farm animals in this country and a short discussion on cyanide poisoning and of the treatment evolved in America of animals so poisoned.

A small number of rabbits and sheep were experimentally poisoned, and of the eight rabbits poisoned three died, but to none of the animals which died was the remedy administered. The remedy was also found to be successful in the treatment of sheep poisoned with KCN.

Our thanks are due to Dr. Mann, of the Molteno Institute for Parasitology in the University, to Sir Joseph Barcroft for much helpful advice and discussion, and to Professor Dalling, then Director of the Institute, for permission to carry out this work.

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THE KIDNEY OF THE INDIAN ELEPHANT
(Elephas indicus)

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A 36-YEAR-OLD female Indian elephant (*Elephas indicus*), weighing 3½ tons, was destroyed in the gardens of the Zoological Society, Edinburgh, 1943.

A post-mortem examination of all the organs except the left kidney was carried out by the staff of the Pathology Department at the Royal (Dick) Veterinary College. The left kidney was removed more or less intact and in a comparatively fresh state. The weight (23 lb.), extent and the abundance of fibrous perirenal tissue made the operation of removal without injury rather difficult. The intention was to investigate the disposition of the ureter, but as the organ was in good condition it was decided to inject the renal artery as well as the ureter. Whether the kidney of the Indian and African elephants have a similar structure, the writer of this note is not competent to judge, having examined only one kidney of an Indian elephant. The inconsistencies of the anatomical descriptions given by the writers on the African elephant's kidney do not give a comprehensive basis on which to form an intelligent comparison. It was not possible to give a description of the kidney on removal from the abdomen without interference, therefore the following details are a simple accurate account of a celluloid cast of the ureter and arteries.

Historical

It will be observed from a review of the available literature that only the kidney of the African elephant has been described. Nellie B. Eales noticed that "the kidney was lobulated and covered by a strong peritoneum. The ureter was divided into calyces which collected from the several lobes."

J. Hyrtl observed "the kidney to resemble that of man in the tubular elongation of the calyces."

W. A. Forbes and A. Von Mojsisovics both mention that "the kidney is lobulated in the young animal but in the adult animal exhibited only 'fields.'"

Max Weber considered "the kidney to be non-lobulated."

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W. Donitz probably gives the best and longest detailed account of the kidney. He contended that " the kidney was divided into ten lobes little marked on the surface. The lobes were formed by a strong connective tissue passing through the organ. Each renculus appeared to be independent. The blood system, however, is common to all the lobes or each lobe interconnected. There are no papillæ at the pyramids. The ducts of Bellini form one large tube (*Tubus maximus*), which opens on the blind end of the calyx."

A general fundamental plan of the renal arterial circulation in many mammals is described by Duncan M. Morrison and his co-workers.

Technique

The celluloid corrosion method is well enough known and in consequence does not require repetition. The pressure used varied between 300-600 mm. of mercury.

The solution injected into the ureter was not coloured, but the solution used for the arteries was tinted red. The manipulation of so large an organ during the corrosion process required considerable care. It was found necessary to give repeated baths of acid followed by careful washing with water from a fine spray until the tissue was completely removed.

Ureter

The ureter enters the kidney at the hilus as a comparatively wide tube and it immediately divides into an anterior and a posterior major calyx. The anterior major calyx gives origin to an intermediate or middle calyx, which, on account of its extent, may be considered as the third or middle major calyx. The diameter of the two major calyces is reduced to half the calibre of the ureter. The anterior major calyx is still further constricted immediately beyond its point of origin. Before the middle major calyx is given off, the anterior major calyx is slightly dilated. Each limb, following this enlargement, is contracted and then gradually increased in bore to form a bulbous swelling from which the next series of branches arise.

Anterior Major Calyx. From the dorsal surface of the dilation on the anterior major calyx, a short minor calyx projects dorsally. The length is 1½ inches with a diameter of 1 inch. The termination of the minor calyx is unusual, in that it forms a central and two lateral recesses. The central recess is broad and convex. The lateral recesses have a horn-like appearance, jutting out from either side of the central recess, and are 1 inch long. Donitz mentions a " Tubus maximus " formed by the ducts of Bellini. These are unquestionably the horn-like recesses of the calyx, which opens on either side of the larger central recess. Most of the renal tubes open into the lateral recesses, giving them a feathery appearance. Not quite so many renal tubes open along the middle portion of the central recess. At the neck of the central recess is a distinct narrow groove, in all probability of vascular origin. From the lower anterior margin of the bulbous swelling a larger and longer tube, the continuation of the major calyx, runs forward 2½ inches to end in a blind central portion and two minor calyces. The calyces are placed medially and laterally, and each is an inch long. The characters of each calyx are similar to the description

given for the proceeding minor calyx, except that the " horns " are not so long.

Middle Major Calyx. A long broad tube (4 in.), which also dilates before the origin of the minor calyces is effected. The dorsal minor calyx is the largest and thickest, terminating in the central and two lateral recesses. The short prolongation of the major calyx ends by giving origin to two short minor calyces, the lateral calyx is a narrow tube ending in the form of a " T." The stroke of the " T " receives the numerous renal ducts. The medial minor calyx is short and stumpy, with two long narrow lateral recesses. The central recesses of these calyces appear to receive a very rich supply of renal tubes, comparable to the supply to the lateral recesses.

Posterior Major Calyx. The posterior major calyx is similar to the other two calyces except that the bulbous dilation is greater. Two dorsal minor calyces spring from the dilation with the prolongation of the major calyx giving two minor calyces, each with similar recesses. Therefore the posterior major calyx has four minor calyces instead of three, as indicated in the others.

Each major calyx is 4 inches in length and the average diameter is an inch.

There are ten minor calyces, six are each approximately an inch long. The remaining four are each no more than half an inch long, with a varying diameter from half an inch to an eighth of an inch.

The middle calyx is almost equidistant from the anterior and posterior calyces, being only slightly nearer the former.

The minor calyces have a definite inclination towards the dorsal surface. The lateral recesses are at right angles to the minor calyx, pointing on one side to the cortex and to the hilus on the other.

The Arterial Blood Supply. The " corrosion method " is of great value in determining the vascular supply of the kidney. The illuminating picture portrayed gives a very clear conception of the larger vessels and the ureter.

From the disposition of these structures the lobation and pyramidal formation of the kidney of the elephant may be established without much difficulty.

A superficial dissection of the tissues surrounding the kidney would have permitted a more complete study of the lobes and given a better view of their topography. This procedure was avoided to ensure that no damage would be done to the organ or the blood supply, and this precaution is of some moment in dealing with so large an organ.

The removal of the kidney from the abdominal cavity did serious damage to some of the perirenal vessels, and in consequence some of the injection was lost and the pressure difficult to maintain. Cut vessels of any size were noted and ligatured, but some of the finer cortical perforating branches which had been severed infiltrated the injected mass into the perirenal tissue. Before the leaking vessels on the under surface had been detected and dealt with some fluid had percolated into the surrounding tissue. In spite of these technical hitches a surprisingly useful cast resulted.

The description which follows is entirely developed from this single cast of the arterial system of the left kidney. Many variations in the branching of the arteries may occur in other kidneys of the same species. The arterial distribution appears to follow a general scheme as evinced by the study of the vessels in the kidneys of most mammals.

XUM

Renal Artery. The main artery enters the kidney at the hilus following the ventral surface of the large ureter. This large artery divided into an anterior and a posterior branch at the site of division of the ureter into two calyces. The bore of these vessels is similar and a little less than that of the parent vessel.

Another large branch breaks away from the anterior artery behind the origin of the middle calyx which it follows. The bore of the middle artery is about the same as that of the now reduced anterior vessel. The posterior vessel retains its original diameter, as might be expected, giving no branches up to this point and having a larger area to supply than either of the above branches.

Anterior Artery. This vessel follows the anterior calyx to beyond the bulbous enlargement, where it gives off an interlobar branch laterally, crossing over to assist in supplying the renal pyramids associated with the middle calyx.

The anterior artery pursues a course for a short distance along the anterior calyx before the next interlobar vessel is given off. This vessel runs medially to the inner renal pyramids. In a short distance the main continuation of the anterior artery splits into two interlobar branches, one running antro-medially and the other antro-laterally. There are, therefore, four interlobar vessels, each giving numerous branches which gain the bases of the pyramids, and here change their direction to become the arcuate vessels.

Middle Artery. This vessel courses straight along the ventral surface of the middle calyx and beyond the central recess before it splits into two branches. A short distance from the termination of the calyx a number of branches are noticed coming from either side of the vessel. These vessels are short and have many short branches, which will supply the adjacent cortical tissue, and no doubt do behave as the other arcuate vessels. The terminal branches reach the bases of the renal pyramids and are followed by the arcuate vessels.

Posterior Artery. This large vessel is associated with the posterior major calyx along its ventral surface. Upon reaching the bulbous enlargement it gives a branch which runs in a posterior direction.

The main artery continues laterally for almost 1 inch, when the trunk splits into two branches. One branch goes medially to assist in the supply of the renal pyramids of the middle calyx. It will be observed that both the anterior artery and the posterior artery each contribute a vessel to the pyramids of the middle calyx. It is not intended to imply that all the rami from these vessels are entirely devoted to these pyramids. On the contrary, each branch gives considerable contribution to the adjacent pyramids of the juxta lobes.

The other branch has a short course until it splits dicotymously, a division crossing the dorsal aspect of the calyx to run in a postero-lateral direction and a division which pursues a lateral course. The distribution and fate of these vessels is as indicated for the terminations of the anterior vessel.

It will be seen that the posterior vessel has four interlobar branches, the same number as the anterior branch.

Arcuate Arteries. These vessels, as the term indicates, describe a curve; the degree of the curve depends on the age of the animal. According to Morrison, the "arching" is more marked as age advances. The convexity of

XUM

the arch faces the outer surface of the organ. From the convexity of the arch many small interlobular rami radiate through the cortical tissue, and from these will arise the afferent vessels and glomeruli. In this corrosion preparation these latter finer divisions are not injected. It is, however, a fair assumption that the general plan of distribution will be affected in the kidney of the elephant as it is in most of the mammals.

The arcuate vessels usually proceed to ramify into smaller vessels which have a parallel course to the surface and are not far removed from the same plane as that of the parent. There does not seem to be any definite design of termination, but quite a variety of endings. The artery is gradually becoming smaller as the interlobular aboresence is detached. The final ramification is into one, two, three or more interlobular vessels.

All the interlobular rami appear from the convex side of the artery with the exception of a few short abortive rami from the concave side. These branches do not show any indication of branching and may end in a glomerulus or the immediate tissue of the parent vessel. All the arcuate vessels do not appear to behave in the same manner. Some of the arcuate vessels run almost vertically through the cortex, penetrating the thin capsule, to ramify in the neighbouring perirenal tissue. In their passage through the cortex they adhere to the general plan and supply interlobular branches. The perforating capsular vessel on gaining exit from the kidney forms a rich plexus of small vessels, covering an extensive area for so small a vessel.

The minor arcuate vessels are mostly short and their ramifications not so profuse. Their interlobular branches are not so long nor do they give so many afferents.

Interlobular Arteries. These vessels do not appear to have any definite scheme of origin. They arise from the arcuate vessels and as shown from the convex surface. The interlobular arteries branch from the parent trunk at varying angles. The vessels arising from the early part of the arcuate artery come away at a wider angle than do the later vessels. Generally, the early vessels are strong and longer and have more branches.

The interlobular arteries frequently ramify into several sub-divisions and these rami may show further ramifications. All the divisions give minute afferent glomerular twigs. A common characteristic is the V division of the terminal interlobular branches.

When the interlobular artery breaks up into several branches, whether the original trunk is long or short it gives few afferent glomerular twigs.

The afferent glomerular twigs arise from the interlobular arteries and its sub-divisions in no apparent order. These short and fine twigs are varying, numerous, and appear at intervals from the circumference of the vessel. They are mostly at right angles to the parent vessel, except the terminals, which form an acute angle. It is frequently observed that the interlobular arteries terminate by piercing the capsule of the kidney and ramify in the perirenal tissue.

It will be noted that the arterial tree of the elephant's kidney does not differ in any degree from the generally accepted conception. The renal artery and its early branches are bigger than in most animals.

The finer divisions compare favourably with those divisions in other animals.

XUM

Summary

(1) The kidney is lobulated and multipyramidal.

(2) The ureter branches into calyces. The major calyces dilate to form bulbous enlargements. The minor calyces terminate in recesses from which radiate the renal collecting tubes.

(3) The arterial tree is in general similar to the plan of the arterial distribution in the kidneys of mammals.

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PHENOTHIAZINE AS AN ANTHELMINTIC IN HORSES

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PIONEER workers[7, 12, 15, 19, 20, 27] with phenothiazine as an equine anthelmintic set a tentative dose rate of 70 to 90 grams/1,000 lb ; however, it soon became apparent that smaller doses were adequate. It is now generally agreed that phenothiazine in doses of 30 grams/1,000 lb. is the most efficient anthelmintic yet available for removing the various species of strongyles from the cæcum and colon of the horse. The drug has achieved widespread popularity for this purpose and many thousands of horses have been treated with highly satisfactory results. Eventually reports appeared in veterinary literature of anæmia, illness and a few fatalities following the use of phenothiazine in horses. It is the purpose of this paper to discuss the toxicology of phenothiazine as an equine anthelmintic.

Review of Literature

To date there are reports in the literature of 22 experimental horses which have become sick and of which five have either died or been killed following phenothiazine therapy[2, 7, 8, 9, 12, 20, 21, 26, 27, 30], and of 90 field cases of suspected phenothiazine poisoning with 32 deaths[6, 9, 10, 14, 18, 22, 23, 28, 29, 30]. Although the majority of these reports fail to include such highly important facts in the history as previous diet, weight, age, condition, etc., an analysis of the available data reveals some interesting features. The most significant finding is that of 97 head treated in the 12 outbreaks, 90 became sick and at least 32 died. Obviously either the particular lot of drug used contained some toxic impurity or some environmental factor was involved. The differences existing in the colour of phenothiazine obtained from various distributors can be explained on the basis of oxidation reactions, and several samples which have differed in appearance have all been found to be of equal purity[16]. Further, it has been

noted that losses may occur in one group of horses treated with a certain lot, whereas others treated from the same lot will respond satisfactorily[9].

On the other hand, there is much to support the belief that some environmental factor is involved in suspected cases of phenothiazine poisoning. The very fact that in nearly all reported cases the vast majority of treated horses have been affected rules out idiosyncrasy and individual sensitivity or susceptibility and points to the operation of an environmental factor. Evidently breed and age can be eliminated as factors, for reports have been received of suspected cases in horses of nearly all breeds and all ages from six months to aged. An obvious explanation is that excessive doses have been employed. In the majority of cases this is true, although Taylor[28] reported seven cases in which recommended dose rates or even lower had been used. Again, the early investigators used doses two to three times as large as those used at present and reported no ill-effects. Evidently excessive dosage is not the sole factor involved, although it undoubtedly may be a determining factor.

Fincher and Gibbons[9] are the only observers who adequately describe the condition of their cases prior to treatment. They note that the majority were in very poor condition, constipated, emaciated and anæmic. Others refer simply to the cases as being in fine or good condition, or omit any reference to this point. Some of the descriptions of pre-treatment conditions are apt to be misleading; for example, Schmidt *et al.*[23] speak of four yearling colts weighing about 375 lb. each, and describe the general condition of their subjects as not being "poor, weak or suffering from infectious anæmia." One assumes they were in good condition, yet a 375 lb. yearling of any breed can hardly be in excellent condition. Nothing is said in any but Fincher and Gibbons'[9] report of the condition of the fæces, degree of parasitism, or previous digestive disturbances.

There is but meagre information in regard to diet and environment previous to treatment, including any sudden or radical changes. Of the 15 reported field cases, nine fail to mention either pre-treatment diet or environment. Those cases in which the previous diet is given reveal but a single factor which might offer a logical approach. The cases reported by Fincher and Gibbons[9] were being underfed on poor quality hay and had very limited exercise; those reported by Jones and Kelso[18] ate straw and manure from a pig pen; and in several cases there was undoubtedly an abrupt radical change of feed. For example, Hatcher's[14] cases were just back from the races in fine condition and were given 2½ lb. of a mixture of oats 90, bran 80, molasses 40, and phenothiazine 25 compressed into pellets. In these cases the possibility of indigestion due to feed conditions is certainly present. The method of administration is apparently not a factor, since cases have been reported following every known method of administration except small repeated doses.

Symptoms and Pathological Anatomy

Symptoms and autopsy findings reveal a confusing picture. Of 82 field cases in which symptoms were recorded, 52 have shown anorexia, 48 anæmia, 40 weakness, 29 icterus, 27 colic, 23 scanty urination, 21 constipation, 20 increased temperature for the first week, and 19 a fast pulse. Seven fatal cases

XUM

were autopsied, and of these five showed enlarged kidneys and spleen, three enlarged heart, two enlarged liver, and one impaction of the colon, one necrosis of the colon, one nephritis, one nephrosis, etc. On the other hand, of 21 experimental cases, 13 showed anorexia, 10 colic, seven constipation, six malaise, six icterus, five hæmoglobinuria, four frequent urination, four reduced peristalsis, two increased temperature and two fast pulse. One died and three were killed, and of these two showed enlarged kidneys, two congestion of the small intestine, and one icterus, one constipation, one petechiæ on the kidney, one mucosal necrosis of the kidney, one congested liver, one congested kidney, and one congested colon.

Errington and Westerfield[7] were the first to demonstrate the presence of anæmia following phenothiazine therapy. They demonstrated that doses of 60 to 90 gms. for thoroughbred yearlings caused a decrease in red blood cell counts of about 3,000,000/c.c. in six to 13 days, and spontaneous recovery followed in a month. They also showed that therapeutic doses of 30 grams could be given with no significant effect on the blood picture. Later Errington[8] confirmed his previous findings and concluded that doses of 30 grams for thoroughbreds are effective as anthelmintics and have no deleterious effect on the red cells. This conclusion has been repeatedly confirmed by a number of investigators[13, 16, 26] and it can now be rather definitely stated that anæmia, severe enough to produce clinical evidence, is only apt to occur when the dosage exceeds the accepted rate of 30 gm./1,000 lb. Swales *et al.*[26] have given rather convincing evidence of this in one of their experimental horses. Following a dose of 70 gm./1,250 lb., a decrease in the red cell count of 2,400,000 was observed in 20 days, but after a rest of 97 days a 30 gram dose produced but little change in the blood picture.

Collier and Allen[4] have demonstrated that hæmolysis of horse red cells by saponin or lysolecithin is powerfully accelerated *in vitro* in the presence of phenothiazone and potassium leucophenothiazone sulphate. Neither oxidation product had any direct hæmolysing effect on horse erythrocytes when tested alone. Lysolecithin is believed to be a normal lysin in mammalian circulation, and Swales *et al.*[26] have shown that potassium leucophenothiazone sulphate is probably the derivative of phenothiazine in horse blood. Obviously the inference is that in relatively high concentrations of the conjugate the normal activity of lysolecithin is accelerated and hæmolytic anæmia results.

Discussion

A critical review of symptoms and autopsy findings in suspected experimental and field cases of phenothiazine poisoning in horses reveals some interesting findings. Apparently, phenothiazine rather than any of its oxidation products is the active anthelmintic principle[6, 11, 16, 26], whereas the absorption of the oxidation products account for the vast majority of the symptoms of poisoning. Collier *et al.*[5] believe that oxidation and absorption take place largely in the cæcum of the horse. If this be true, any factor which increases oxidation and absorption will serve to decrease the anthelmintic activity, increase the concentration of leucophenothiazone sulphate in the circulation and increase the toxic effects. Experimental evidence[5] has shown that micronised phenothiazine in

FIG. 1

A. Ureter.
B. Anterior Major Calyx.
C. Medial Major Calyx.
D. Posterior Major Calyx.
E. Minor Calyx.
F. Central Recess.
G. Lateral Recess.
H. Uriniferous Tubules.

XUM

FIG. 2

A. Renal Artery.
B. Anterior Branch of Renal Artery.
C. Medial Branch of Renal Artery.
D. Posterior Branch of Renal Artery.
E. Interlobar Artery.
F. Arcuate Artery.
G. Interlobular Arteries.
H. Branches Perforating the Capsule.
I. Ureter.
J. Bulbous Enlargement of Calyx.

XUM

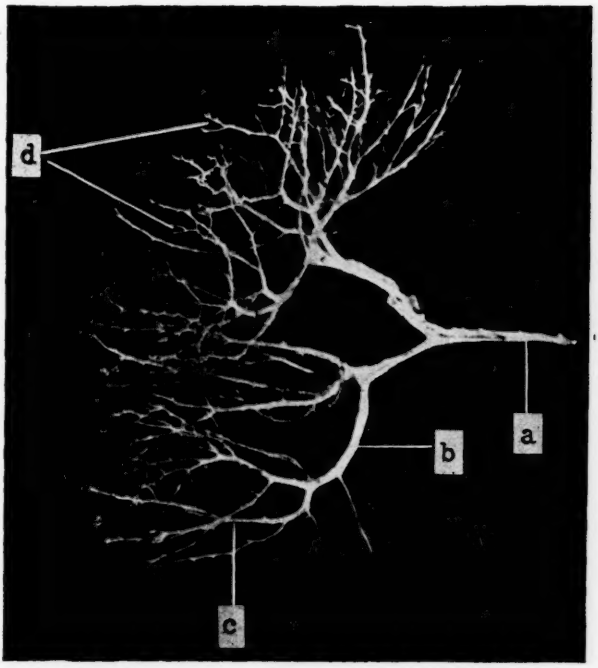

FIG. 3

A. Interlobar Artery.
B. Arcuate Artery.
C. Interlobular Artery.
D. Terminal Branches of Interlobular Arteries.

XUM

which 80 per cent. of the particles were less than 4 microns in diameter, is 90 per cent. oxidised and absorbed, to be eliminated in the urine, whereas ordinary coarse commercial phenothiazine was only a little more than 50 per cent. oxidised and absorbed. The size of the dose is likewise a determining factor in the amount of conjugate found in the blood-stream, the smaller the dose the lower the concentration of potassium leucophenothiazone sulphate in the serum. Both of these statements have been confirmed by experimental evidence[5, 26]. Theoretically, any factor which tends to diminish peristalsis could conceivably increase both oxidation and absorption. Although experimental confirmation is lacking, there are strong indications from symptoms and autopsies of suspected cases of phenothiazine poisoning to support this view. As early as 1940 Swales[24] recognised that phenothiazine in itself is inclined to cause constipation, and he states "unless the particles of phenothiazine move through the tract fairly rapidly they are unduly exposed to oxidation and consequent absorption from the upper parts of the alimentary canal. Under such circumstances a very large proportion (i.e., over 80 per cent.) may be absorbed and excreted through the urinary tract."

A careful examination of the reported symptoms reveals constipation, icterus, reduced peristalsis, colic and frequent scanty urination which are often associated with impaction of the colon in the horse. Further, many report prompt recovery following blood transfusions[9, 23, 10], which might be assumed to combat not only the anæmia, but also the weakness and shock resulting from severe constipation. Fincher and Gibbons'[9] cases were constipated before treatment, Jones and Kelso's[18] were eating from a straw and manure pile, Roberts'[21] were fed on chopped alfalfa, and Boley *et al.*[2] report a case in which the horse had no feed for five days prior to treatment. In these cases, at least, the opportunity for digestic disorders was present. The high incidence of colic, weakness, abdominal pain, constipation and restlessness indicates indigestion in the nature of impaction of the colon which would undoubtedly increase both oxidation and absorption. In addition, the majority of cases have followed excessive doses. In this connection it is of interest to note that Taylor and Sanderson[27], Grahame *et al.*[12], Lapage[20] and other early workers administered their large doses only after thorough preparation of the horses by two or more warm bran mashes.

Work in California[16, 17] has indicated that the small repeated dose method of administration is a safe and satisfactory method of treating poor, weak or anæmic horses (usually cases of primary strongylosis), brood mares and weanlings. It is possible that very little absorption takes place with this method. At any rate, when the daily dose is 3 grams or smaller, no staining of the urine is observed. In emaciated horses this method has been used, giving 1-3 gms. of phenothiazine mixed with powdered blood and digestive stimulants, with very satisfactory results. No anæmia has been observed in 28 head so treated. Because of the constipating effect of the drug, the author has routinely recommended thorough preparation of horses with several bran mashes before treatment and administration of a single dose in a bran mash in all cases other than those where small repeated doses are indicated. Where this routine has been followed, no cases of suspected phenothiazine poisoning have been observed.

XUM

Summary

The available literature on the use of phenothiazine as an equine anthelmintic has been reviewed, with special emphasis on toxicology. Apparently increased oxidation and absorption of the phenothiazine derivatives accounts for the observed symptoms. Phenothiazine poisoning can be kept at a minimum by employing doses no larger than 30 gm./1,000 lb., thorough preparation of the horse with several bran mashes prior to treatment, and the use of small repeated doses in weak emaciated anæmic horses.

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Abstract of Current Literature

SECOND REPORT OF THE COMMITTEE ON VETERINARY EDUCATION IN GREAT BRITAIN*

THE Committee, under the chairmanship of Dr. T. Loveday, M.A., Vice-Chancellor of the University of Bristol, has recently issued its second report. It will be recalled that the first report was issued in July, 1938. The Committee were invited by the Secretary of State for Scotland and the Minister of Agriculture and Fisheries to review their earlier recommendations. In a letter from the Minister of Agriculture and Fisheries the attention of the Committee is drawn to the intention of the Government of "maintaining after the war a healthy and well-balanced agriculture as an essential and permanent feature of national policy" and to the "need of adequate attention for maintaining the health of our livestock, particularly our dairy herds." It is also stated in the letter that "We are satisfied, therefore, that in the

* Cmd. 6517, H.M. Stationery Office, price 6d. net.

changed circumstances not only is a better and in fact the best possible training for the veterinarians of the future of vital importance to agriculture, but also numbers substantially greater than exist at present or have hitherto been envisaged will be required for the efficiency and well-being of the livestock branch of the industry."

Some of the principal conclusions and recommendations in the report are as follows:—

The Ministry of Agriculture and Fisheries expect that there will be a demand for about 220 veterinarians annually for a period of roughly ten years, with a possible reduction to 150 annually thereafter. The Committee do not consider that the estimate of 220 is excessive and have framed their recommendations on this basis, but they see no reason to believe that the demand will fall after a decade to 150.

The Committee are of opinion that the annual output of 220 graduates can best be met, in the interests of students and teachers, by adding two new training centres, one at Cambridge and the other at Bristol. The output from the respective centres should be about 30 from Liverpool, Glasgow, Cambridge and Bristol, and 100 from London and Edinburgh together.

The Committee are convinced that the best possible training for veterinary surgeons can only be secured by persuading Universities to undertake responsibility for the training.

Veterinary studies should be given full University status, a degree in veterinary medicine becoming a registrable qualification.

The Council of the Royal College of Veterinary Surgeons, altered in constitution but with enlarged duties, should continue to maintain a minimum standard of qualification and should remain the governing body of the profession.

The Committee recommend in respect of Universities authorised to confer registrable qualifications (1) that legislative provision should be made in respect of veterinary education identical *mutatis mutandis* with that already provided in respect of medical education, the Council R.C.V.S. being given corresponding powers to those of the General Medical Council; (2) that the Council R.C.V.S. should be required to place upon the register of members of the College the names of all graduates of Universities authorised by the Privy Council to confer degrees which are registrable qualifications; (3) that the Council should, in mutual agreement with a University, inspect the premises and equipment used for veterinary education and support any representations by the University with regard to deficiencies therein or in staffing.

In respect of veterinary schools not being Universities, the Committee recommend that the Council R.C.V.S. should be empowered (1) to inspect such schools and if the instruction given therein is found insufficient, that it should have the right to require improvement of the instruction, and if this is not effected to report the matter to the Privy Council; (2) to give support, when thought advisable, to representations by the authorities of a

XUM

school similar to the support given to a University under (3) above in respect of premises, equipment and staffing.

The Council of the R.C.V.S. should consist (apart from members from Eire) of 33 persons—17 to be elected as at present, 12 to be members of the R.C.V.S. appointed by Universities and affiliated schools (two each by the Universities of Cambridge, Glasgow, Liverpool and Bristol; one each by the Universities of London and Edinburgh and by the Royal Veterinary College and the Royal [Dick] Veterinary College), and four appointed by the Crown who need not be members of the R.C.V.S. This means that of the 33 members, at least 29 would be members of the R.C.V.S.

The Council of the R.C.V.S. should take power to employ teachers in the schools and members of Council as external examiners for the Diploma.

The Committee strongly urge any University given the power to confer registrable degrees to include practising veterinarians among their external examiners in clinical subjects and to consult the Council of the R.C.V.S. in selecting such examiners.

The R.C.V.S. should continue to be responsible for the conduct of the examinations (other than those for University degrees) for the diploma of membership of the College, the Fellowship diploma and the diploma in veterinary state medicine.

If the Committee's recommendations are accepted membership of the R.C.V.S. would still be the licence to practise—membership would be acquired either by the present method or by obtaining a registrable degree at a University authorised by the Privy Council to grant such a degree.

A field station should be attached to each teaching centre and an adequately equipped and staffed hospital for large animals should be provided.

The Committee repeat their former recommendations that a pupilage of at least six months on a farm at varying seasons of the year should be required and that every student should spend at least six months with a veterinary practitioner, at least half of the period during the fourth and fifth years of study.

The carrying out of research is an essential function of all teaching institutions, and provision should be made for funds for this purpose.

Bursaries should be provided, so that no boy or girl whom a training centre is prepared to accept shall be precluded from training by lack of means.

Graduate scholarships should be made available to enable students of outstanding ability to work for higher degrees after qualification.

Private endowment for veterinary education is recommended.

The Committee favour the idea that a faculty of veterinary medicine should be established in a University at which there is a training centre.

The institution of an inquiry on the subject of unqualified practice is of urgent importance.

The Committee estimate very roughly that the acceptance of their proposals would involve a capital expenditure of the order of two million pounds and an additional recurring expenditure of about £210,000 a year.

XUM